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OBJECTIONS

TO SOME OF THE RECENT VIEWS UPON THE

PATHOLOGY OF TUBERCLE

AND

PULMONARY CONSUMPTION,

BEING THE

Address in Medicine read before the Canadian Medical Association,

ON THE 5th AUGUST.

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OBJECTIONS *to some of the recent views upon the Pathology of Tubercle and Pulmonary Consumption, being the Address in Medicine read before the Canadian Medical Association, on the 5th August.* By R. P. HOWARD, M.D., L.R.C.S.E., Professor of Theory and Practice of Medicine, McGill College.

Gentlemen of the Canadian Medical Association:

As you have generously left to my own choice the subject in medicine upon which I am to address you, I have selected one which because of its great importance and of the differences of opinion respecting it that exist among the leading minds of our profession, seems worthy of the occasion.

I ask your attention and indulgence while offering some observations upon the remarkable views that have been recently propounded upon the Pathology of Tubercle and Pulmonary Phthisis.

Perhaps the most startling pathological doctrine advanced in modern times on respectable authority is that miliary tubercle and so-called tuberculous infiltration are due to the absorption of the *caseous* detritus of the products of some pre-existing local disease as a pleurisy, pneumonia, scrofulous gland, diseased bone, abscess, fistula, etc.,—that tuberculosis is *either* an *absorption* disease nearly allied to pyæmia (Waldenburg) or a specific *infectious* disease like small pox (Burdon-Sanderson).

More or less closely connected with this new doctrine, but not necessarily arising out of it, is the old thesis revived in a modified form, and because of his able advo-

cacy of it, usually associated with Niemeyer's name, to the effect that the ordinary form of chronic phthisis pulmonum is of inflammatory origin and due to a so-called "caseous pneumonia," which may be induced by a *catarrhal* bronchitis or the local irritation of blood poured into the bronchial cells in pulmonary hemorrhage.

As these views are of overwhelming importance, in view of the practical consequences which must inevitably follow their establishment, I venture to raise some objections to them for the consideration of the members of this Association.

The *absorption* theory of tubercle rests mainly upon the interesting experiments initiated by Villemin in 1865 and subsequently confirmed and extended by Andrew Clarke, Burdon-Sanderson, Wilson Fox, Waldenburgh, Cohnheim, and others. These experiments revealed the important fact that in the Guinea pig, and in some other animals, the inoculation of tubercle, pus, putrid muscle, etc., of tubercle which had lain several months in alcohol, or had been submitted to the action of fuming nitric acid, or of carbolic acid, will produce *primary* lesions at the site of inoculation and *secondary* lesions in the internal organs, which appear to be identical with tubercle. Even the local irritation of a seton of cotton or of silver wire will produce similar effects in the Guinea pig.

Now, lest these inoculation experiments upon animals should be assumed to have proved more than they have—let it be borne in mind (1) that every animal has its own special organization and probably its own special aptitudes as regards diseased action; and (2) that it has yet to be shown that the inoculation of tubercle or other material is capable of producing lesions identical with tubercle in the several organs of the human body.

(3.) Clinical experience does not show that the irritation of setons or issues is causative of tuberculous disease in man.

(4.) Local suppuration when productive of secondary

remote lesions in various parts of the human body tends to develop pyæmia, with its peculiar metastatic deposits, or amyloid disease of the viscera—not, I believe, tuberculous disease. I regard this fact as strongly opposed to the inoculation doctrine. The very condition, chronic suppuration, as seen in necrosis, or caries for example, and which resembles so closely the action of a seton, is the very condition which is recognized as *the* cause of amyloid disease of the liver, spleen and other organs.

(5.) It is asserted that pleurisy and especially chronic pleurisy frequently causes consumption.

Now Dr. Blakiston watched for some years the course of 79 cases of chronic pleurisy with the following results — 10 were lost sight of — of 16 it was only known that they were living — and of the remaining 53 *not one* had become phthisical. Dr. Payne-Cotton's experience and Dr. Flint's is opposed to the view in question and coincide with Blakiston's. M. Aran and M. Siredey both contend that the autopsies of empyemic subjects show that tubercle is more frequently absent than one might expect.

Dr. A. Attimont's researches give 80 definite cures out of 130 cases of empyema, many of which moreover had been watched for a long time. And in 29 autopsies of empyema tubercles were absent 20 times and present only 9 times. If so many persons recovered perfectly from empyema, and if tubercle was found but 9 times in 29 cases, it certainly does not appear probable that empyema can be a very frequent cause of pulmonary tubercle.

When tubercle appears to have followed an empyema or a pleurisy, several explanations may reasonably be offered of the relationship: Tubercles may have existed in the lungs or pleura at the time of the invasion of the inflammation — or, as was admitted by Trousseau, a predisposition may have existed, which the local inflammation developed into actual disease.

(6.) If there are not sufficient grounds for asserting that the absorption of the products of pleurisy causes the

formation of tubercle, what about other local inflammatory affections?

Much importance cannot be reasonably attached to the statement of Troltsch, that purulent otitis not rarely precedes tuberculous meningitis, and even general tuberculosis, when we reflect how common an affection otorrhœa is in childhood.

(7.) It has been claimed that *fistula in ano* may initiate tubercle. But let it be noted that according to Pollock, and such has been my own experience, "the phthisis is the earliest affection," although it "occasionally happens that the fistula precedes all symptoms of consumption." Indeed, the same authority remarks that "in the larger proportion of cases it [the fistula] is found in the *third* stage, next to this in the second.

Now, if the absorption of corpuscular products from abscesses, ulcers, etc., may induce tuberculous disease, how comes it that a *fistula in ano* is, according to Pollock *never associated* with *acute* phthisis? the very variety in which the tubercle par excellence (miliary tubercle) of Virchow is present? And that the existence of anal fistula in chronic consumption appears to *prolong* the duration of the pulmonary disease, instead of causing it to extend and take an active course through the constant absorption of the inflammatory products?

(8.) Disease of the *bones* is regarded as one of the pathological conditions likely to produce tuberculous disease.

To show that disease of the bone is far from constantly or even frequently a cause of consumption, I would cite M. Coulon, who in 130 children suffering from scrofulous disease of bone found only three that had phthisis.

Mr. J. W. Hayward, writing upon another subject, gives some facts which appear to me corroborative of the view I am defending. "Of 85 consecutive cases admitted into the hospital for sick children for various tubercular affections, in only *one* was there any bone or joint disease."

"Of 134 cases of chronic bone or joint disease of which" he "took notes in the Hospital for Sick Children, in only 9 was there any sign of tuberculosis, and but 17 displayed other signs of scrofula."

Of 790 cases of *bone* and *joint* disease in adults and children admitted into St. George's Hospital, disease existed in other parts in 80 instances only, or in about ten per cent., and it may be safely assumed that the co-existing disease was not in every instance tuberculous.

(9.) There is one affection especially characterized by a tendency to caseous degeneration of the lymphatic glands which because of its frequency ought, if the absorption thereby be true, to be found very frequently associated with consumption. What are the facts?

Mr. B. Phillips taking enlarged glands with sinuous ulcerations and enlarged joints as diagnostic of scrofula, found scars resulting from scrofulous abcesses in only 7 out of 352 cases of phthisis. Dr. E. Smith, in 1000 cases of consumption, found that only 12.8 % (not 13 per cent.) had suffered from enlarged glands.

Of 1973 cases examined at Brompton Hospital for consumption only 3.8 % (not 4 per cent.) had scrofula.

Dr. Flint writes, "I have collected a number of cases in which young and middle aged persons presenting the characteristic cicatrices on the neck were free from tuberculous disease of the lungs; and on the other hand, it is extremely rare to find these cicatrices in persons who are affected with pulmonary tuberculosis." Such also is my own experience.

But the supporters of the doctrine that tubercle results from the absorption of caseous products rely also very much upon the fact that the able advocate of the theory, Fuhle, having carefully examined 23 cases of acute tuberculosis found *in all but one* or *two* masses of caseous matter, which he assumes to have *preceded* the tubercle.

In reply it may be urged that Wilson Fox has always found the histological elements of tubercle in the walls of

the air cells when caseation was present. That Dr. Schüppel, writing in 1872 upon tuberculous disease of the lymphatic glands, concluded that in all cases, caseous scrofulous glands are the seat of *true tubercles*, and that the caseation is due to the necrosis of the tubercle." That Dr. L. Thaon, writing in 1873, throws great doubt on the existence of any true cheesy inflammation independently of the tuberculous granulations, and concludes that cheesy inflammations are as much manifestations of tuberculosis as the grey granulations (miliary tubercles) themselves; a conclusion almost the same as that entertained by Wilson Fox. And that quite recently, Dr. Friedlander has stated that "though caseous deposits *are* found in *nearly* all cases of disseminated tuberculosis; *in some* a most careful examination has failed to detect them. And moreover, such deposits are so common that they are found in the bodies of 50 % of all adults who have died from any cause. And though the proportion of such deposits is much greater in cases of disseminated tuberculosis, it must be remembered that a very large number of these deposits are actually the *result* of local tuberculosis, all caseous glands for example; * * * * * * "and that we have quite as much right to consider that certain forms of local tuberculosis dispose, even after their relative healing, to the origination of disseminated tuberculosis, as to assume with Buhl that this is due to caseous infection."

Having in my own opinion, shown that the Absorption, theory of the production of tubercle suggestive by the inoculation experiments upon animals is not borne out by clinical experience, nor rendered probable by unanimity of opinion amongst histological authorities, I turn to the other doctrine that the ordinary form of chronic plumonary consumption is of inflammatory origin and due to pneumonia.

This is indeed a question beset with difficulties and upon which the ablest pathologists take sides, and I therefore claim your indulgence while venturing to speak upon the subject.

Most clinical physicians will admit, I doubt not, that ordinary acute (croupal) pneumonia in the great majority of cases runs a short course, its products are completely removed, and no ulterior damage follows. It is admitted that occasionally, though but rarely, this form of pneumonia induces abscess or even gangrene of the organ, but even then the disease can be distinguished from ordinary chronic phthisis.

But it is *catarrhal* pneumonia that the new school of pathologists, ably represented by Niemeyer, Waldenburg, Buhl, and others assert, originate and constitute the most frequent form of chronic consumption, and that miliary tubercle has nothing to do with it and is very frequently absent.

Now I submit that this question cannot in the present state of conflicting opinions and observations amongst histologists, besettled upon histological evidence. Need I remind you of the discordant views expressed by the able men who took part in the debate upon tubercle in the Pathological Society of London last year? The out-come of that discussion was, in my opinion, that the *microscope* does not enable us to distinguish tubercle from the products of lobular pneumonia and from other products which may closely resemble it. Nay, histologists are not agreed as to the histological characters of tubercle itself; and I have before mentioned that they differ as to the relationship existing between tubercle and caseous masses in the lungs; some contending that caseous material may exist without tubercle, but that tubercle results from the absorption of caseous products, — others that wherever caseous materials are present in the lungs tubercle coexists; and others, that caseous inflammations are as much manifestations of tuberculosis as miliary tubercle itself.

Histological evidence then being unsatisfactory we appeal to *clinical*.

After what has been said, it may be asserted with confidence, that in a given case unless the physical signs, the

symptoms, and the whole clinical history during life were those of catarrhal pneumonia, the existence of caseous masses in the lungs could not fairly be taken as proof that the disease was pneumoniac and not tuberculous.

Now the clinical features of catarrhal pneumonia differ from those of ordinary chronic phthisis.

Thus, (*a*) Rokitansky says that catarrhal pneumonia affects children chiefly and is "of rare occurrence in adult life," and Niemeyer himself observes that "we may very properly call it a disease of childhood." The converse is true of chronic phthisis; it but rarely occurs in children, it obtains pre-eminently in adult life.

(*b.*) The chronic form of catarrhal pneumonia is so infrequent that Buhl has lately questioned its existence, and it is met with chiefly, almost solely, in connexion with measles, hooping cough and capillary bronchitis. The same cannot be affirmed of chronic phthisis.

(*c.*) While the statement that a well developed paroxysmal cough accompanied in a few days by expectoration, or at least by the evidence of considerable secretion from the broncho-pulmonary membrane, and plainly explained by the physical signs of bronchitis, is almost invariably true of catarrhal-pneumonia—the statement that short cough or a mere hem, unaccompanied by expectoration for weeks, and either not accounted for by physical signs at all, or by physical signs differing in several respects from those of bronchitis and catarrhal pneumonia, is equally true as a general rule of chronic phthisis.

(*d.*) The physical signs of chronic consumption in the early stage are not those of catarrhal pneumonia. Thus in the former there shall exist for weeks and even months simply feeble respiration or harsh respiration with prolonged expiration and a shade of diminished percussion resonance at the apex of one lung without either whistling or bubbling râles — indeed these may never ensue; and the other apex and the bases of the lungs shall present no signs of disease. Broncho-pneumonia on the other hand, is pre-

ceded by the physical signs of bronchitis — the signs are distributed more or less symmetrically over both lungs, but predominate at the bases of those organs.

In view of the fact that histological examination cannot be relied upon, and that clinical evidence does not support the statement that either croupal or catarrhal pneumonia is apt to terminate in or constitute chronic phthisis, it may be concluded that Niemeyer was wrong in maintaining that chronic phthisis usually originates in inflammation of the lungs.

Admitting that ordinary lobar pneumonia sometimes ends in softening and ulceration of the lungs; that chronic bronchitis sometimes becomes complicated with induration, and that the indurated portions may slough or ulcerate; and that catarrhal pneumonia sometimes is followed by ulcerative destruction of the lungs, yet these issues are so infrequent relatively to the frequency of lobar pneumonia, chronic bronchitis and catarrhal pneumonia respectively, that they cannot reasonably be regarded otherwise than as *exceptional*, and not as the natural course of these affections.

The great fact which nearly all pathologists admit in some form, that a predisposition to pulmonary consumption, inherited or acquired exists, and which has led to the disease being placed amongst the constitutional affections, seems to prove that there is something special and peculiar to the disease which distinguishes it from simple inflammation of the lungs, whether croupal or catarrhal.

Burdon-Sanderson, while applying the facts of animal inoculation to the pathology of consumption in man, admits this *latent phthisical* bias. Virchow and his followers, including Niemeyer, admit that the predisposition to the so-called "caseous" or "scrofulous" pneumonia which the latter regards as the nature of most cases of chronic consumption, is "inherited" as "a vulnerable constitution."

It is this bias or a tendency in the individual that conditionates the peculiar characters and course of the hyper-

plastic or the inflammatory process, whichever it is, that produces consumption — and its recognition is equivalent to the admission that the so-called "caseous" or "scrofulous pneumonia" of ordinary phthisis is peculiar and essentially different from pneumonia occurring in persons free from the inherited or acquired tendency in question. It is this inherited, or perhaps acquired, mode of vital action — this constitutional bias — that causes a bronchitis or a pneumonia to take on peculiarities which distinguish it from ordinary bronchitis or pneumonia. The bronchitis or pneumonia becomes the agency of developing the latent tendency in the individual.

But I must stop lest I overtax your patience. I have attempted to show that it has not been proved by an appeal either to histology or to clinical observation, that tubercle or consumption may be produced in the human subject by the absorption of caseous and other products of inflammation, as it appears capable of in rabbits and other animals. And I have also attempted to show that clinical observation is opposed to the doctrine that ordinary chronic pulmonary consumption consists of simple pneumonia, either croupal or catarrhal, and that if the local process is inflammatory it is at least of a peculiar or specific kind, and to be designated by a distinctive name such as "caseous," or "scrofulous," or tuberculous." It has its own symptoms and signs, runs its own peculiar course, recognizes its own causes and pathology and demands its own therapeutics.

But it does not follow that we may neglect or treat as trivial a bronchitis, an intestinal catarrh, a chronic abscess, or a fistula. Nor do I wish to deny that inflammation plays an important rôle in consumption, infiltrating the pulmonary substance in the neighbourhood of the tuberculous disease with materials prone to degenerate, but simply to maintain that the great majority of cases of chronic phthisis are *not* cases of, and do *not originate* in either lobar or broncho-pneumonia.

Montreal, 31*st July*, 1874.

Since this paper was read I have seen a notice of Buhl's recent monograph upon "Inflammation of the Lungs, Tuberculosis, etc.," and have been much interested on observing that, contrary to the teachings of Niemeyer and his school, he states that neither croupous nor catarrhal pneumonia ever gives rise to phthisis. However, he has described a third form of pneumonia not previously mentioned by pathologists, under the name of "Desquamative pneumonia" the highest degree and the commonest form of which he calls "caseous pneumonia," and regards it as constituting one of the commonest forms of phthisis.

Not only does Buhl agree with the writer that neither croupous nor catarrhal pneumonia passes into consumption, but in opposition to Niemeyer he denies that "caseous pneumonia" ever originates in the catarrhal or croupous form, a view which is in harmony with the tenor of this address. Of course it remains to be seen whether pathologists will accept and confirm the existence of a third variety of pneumonia — the "desquamative" — distinct and different from the croupal and catarrhal forms.

Montreal, 19*th Aug.*, 1874.